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Indications For Open-Heart Surgery In Congenital Heart Disease

By JOHN M. VEROSKY, M.D., *El Paso**

The indications for surgical repair of congenital cardiac malformations requiring cardiotomy and extracorporeal circulation change from year to year, and sometimes almost from month to month, as the improvement in surgical techniques and in the mechanical devices gradually decrease the operative risk involved, and as knowledge of the natural life history of the various anomalies grows through continued study.

For example, six years ago, before the days of successful pump oxygenators for total cardiopulmonary bypass, when atrial septal defects were being repaired by closed techniques, such a defect had to be very large to merit consideration for the blind procedure which carried a high mortality and which sometimes resulted in incomplete closure of the defect.

Cardiologists were loath to recommend this procedure unless serious symptoms were present, with a shunt large enough to produce a pulmonary-systemic flow ratio of three or four to one—a truly huge defect. At the present, an atrial defect worthy of closure is one producing a ratio of two to one. In the future, it may well be that even smaller defects will be regarded as indication for repair.

Much Thought

It requires a good deal of thought to decide which child should be operated. First must be considered the deceptive lack of symptoms in many children who are headed for early disaster. It should be remembered that children with congenital cardiac disability differ from individuals with acquired heart disease in that there is no previously normal state for comparison by the child, parents, or observers. If a child has carried a certain level of exercise intolerance since birth, it becomes surprisingly difficult to judge the degree of the intolerance unless it is very great.

Sometimes a true evaluation of the limitation is obtained only retrospectively postoperatively, when the parents note the remarkable change in the child's activity. In contrast, a child developing acute rheumatic fever will more easily note an alteration in fatigability. Furthermore, children more reliably limit themselves to activities which do not cause uncomfortable fatigue or dyspnea.

Without the external drives, goals, and compulsions of adult responsibilities, they do not press themselves to the point of this discomfort (unless similar external influences, such as the rivalry of team sports, are operative.) In valvular pulmonic stenosis, one occasionally sees a vigorous, well-developed young infant, and frequently sees an active older child who has had very little in the

CURRENT THERAPY

*Dr. Verosky this month is substituting for Dr. Jack A. Bernard, regular conductor of this section.

way of symptoms a short while before the onset of lethal right ventricular failure.

Reliability Varies

Second, the reliability of various signs and symptoms in predicting future difficulties varies with the particular malformation present. For instance, radiologic heart size is a good judge of the size of the shunt in an atrial defect; the larger the abnormal flow, the greater the degree of right atrial and ventricular dilatation.

On the other hand, in pulmonic stenosis with intact ventricular septum, the damaging right ventricular hypertrophy is chiefly concentric; great progression of this hypertrophy can occur without correspondingly great change in heart size; more reliable here than the x-ray is the electrocardiogram.

In atrial septal defects, the intensity of the murmur is of little importance; very large defects can produce only a quiet murmur. In pulmonic stenosis, the murmur is similarly of little help, for even mild stenosis can produce a Grade 4 murmur. In these two instances, the murmurs are of little aid in following the patient.

Septal Defect

In a ventricular septal defect, the development of a basal diastolic decrescendo murmur is highly significant, since it may reflect pulmonary regurgitation secondary to pulmonary hypertension. In a case of Tetralogy of Fallot, the factor most often pressing to operation before optimal age is the presence of severe cyanotic spells—a symptom; here, this symptom is of more importance than x-ray or electrocardiographic findings.

The presence of cyanosis per se in one with Tetralogy is of no sinister import; cyanosis developing in a patient with patent ductus arteriosus or valvular pulmonic stenosis is an ominous development indeed.

Thus, to a large extent, which parameters are of greatest importance depend upon the type of malformation present. Correct evaluation of the situation is dependent upon correct clinical diagnosis.

Decision Reviewed

Finally, a decision to delay surgery must be reviewed periodically. Changes in the child's cardiopulmonary physiology occur with the passage of time, and, as noted above, changes in the surgical outlook also occur.

If a complete anatomic and physiologic diagnosis is available, the decisions are relatively simple. Here are current criteria for the most common lesions:

1. Septal defects with left-to-right shunts (atrial septal defects, ventricular septal defects, and foramen primum defects). Operation is recommended if there are serious symptoms, if there is a pulmonary-systemic flow ratio of two or more, or if there is pulmonary hypertension (unless the latter is severe enough to preclude survival of the patient postoperatively).

2. Pulmonic stenosis. Operation is recommended if right ventricular systolic pressure exceeds 90 mm. Hg. If the pressure is between 60 and 90 mm., operation will probably be necessary, but postponement (with careful following) is a reasonable course. If the pressure is below 60 mm., operation is currently not being recommended.

Tetralogy of Fallot

3. Tetralogy of Fallot. According to Keith, isolated examples of longevity "should not be allowed to obscure the plain fact that the average age of survival is twelve years, and that extremely few patients survive 20 years." This malformation is the commonest cyanotic type surviving past young childhood; even before the days of catheterization it had a better than average chance of being correctly diagnosed.

Since most patients with the illness are obviously blue, not many have escaped detection. Thus, it is one type in which rather reliable survival statistics have been collected. (This cannot be said for many other malformations; in the past, pulmonic stenosis has been misdiagnosed as ventricular septal defect, atrial defects have often escaped diagnosis entirely, innocent murmurs have been called ventricular defects, etc.)

With few exceptions, all patients with Tetralogy should be repaired. If possible, operation should be delayed until at least four or five years of age. If severe symptoms demand aleviation between two and four years of age, total repair, utilizing extracorporeal circulation, should be done, although the risk is higher.

Palliative Procedure

If the infant under two has symptoms serious enough to involve risk of death or cerebral dam-

age, operation should also be done; at the present time, however, the risk in the infant group is so great, that a paliative procedure (such as the Blalock subclavian-pulmonary or Potts aortico-pulmonary anastomosis) is probably preferable, aiming at survival until a later date when curative surgery could be performed. The latter plan is taken with reluctance, since reoperation is generally more difficult, and since pulmonary complications secondary to the artificial shunt can occur, both factors compromising the chances for successful total repair.

4. For the sake of completeness, we wish to include the repair of two lesions not requiring cardiotomy and extracorporeal circulation:

   a. Patent ductus arteriosus. All should be operated, for the usual well-known reasons, except if severe pulmonary hypertension (beyond infancy) forbids repair.

   b. Coaractation of the aorta. All should be operated. There is still some doubt that an infantile anastomosis will grow with the aorta in every case; thus, except under unusual circumstances, operation is best delayed until the age of eight or nine years. This is ordinarily quite safe, since complications in this malady are usually delayed until at least early adolescence.

Anatomic and Physiologic Diagnosis

These indications presume a complete anatomic and physiologic diagnosis. Ordinarily, this includes hydraulic data obtained by right heart catheterization. The decision is less certain if such data have not yet been obtained. In general then, the more difficult decision is when to recommend catheterization and other procedures, such as angiocardiography, sometimes necessary for complete investigation.

The factors operating in this decision cannot be stated quite so categorically. However, with all the precautions noted above in mind, one should consider the following:

Factors Listed

1. Failure to gain. This is an important symptom which, if present without other cause, points to a serious malformation. Normal growth does not, however, necessarily mean that the disease is mild.

2. Frequent respiratory infections, especially pneumonia. This is a common warning sign in many of the patients with left-to-right and some with right-to-left shunts.

3. Cardiac failure. This, with certain exceptions, calls for thorough investigation in the near future.

4. Severe cyanotic spells, particularly if frequent, particularly if progressing to syncope or convulsions.

5. Cardiomegaly beyond a certain degree, depending on the malformation in question. This may be an indication when no other exists (such as in atrial septal defect.)

6. Certain electrocardiographic findings, varying from type to type. For example, pulmonic stenosis with right ventricular hypertrophy producing a 20 or higher mm. R wave in lead V1; ventricular septal defect with progressive right ventricular dominance.

7. Evidence of pulmonary hypertension, especially if progressive.

Circumlocution

It is at once obvious that a circumlocution is involved here: wise recommendation for or against catheterization depends on criteria which vary with the type of anomaly, and yet the exact nature of the anomaly is determined only by catheterization! However, careful clinical evaluation will usually provide a sufficiently accurate diagnosis that the decision can be made with fair certainty. In about 90 percent of cases, catheterization is aimed less at actually making the diagnosis and more at physiologic measurement of the magnitude of defects, at uncovering hidden associated anomalies, and at confirming the major diagnosis.

Pulmonary Hypertension Problem

The problem of pulmonary hypertension is so serious, deceptive, and damaging that it is worthy of special digression. In the presence of a septal defect, the direction of the shunt is determined solely by the relative resistances on the two sides of the defect. Systemic resistance is normally many times greater than pulmonary, so that the flow is generally left-to-right.

In the absence of pulmonary stenosis, the most significant feature which can change this is the state of the pulmonary arterioles. Dextraposition of the aorta, per se, is of no importance. Even the size of the defect is not important in determining direction; the only influence of the size of

the hole is that a small hole offers greater resistance to flow than a larger one; this is a neutral factor in determining direction of flow.

Resistance Increases

In a certain percentage of patients with left-to-right shunts, pulmonary resistance increases, as reflected by elevation of pulmonary artery pressure and right ventricular work; as the resistance rises, the left-to-right shunt decreases in volume, becomes a bidirectional shunt, and finally a predominantly right-to-left one.

If the defect is then surgically repaired, some of these patients will die from intractable right ventricular failure, since the safety-valve against extreme pulmonary hypertension has been removed by the surgery. The resistance (and pressure) can be due to vasospasm in the lungs, to actual arteriosclerotic-like anatomic changes in the vessels, or to both.

At catheterization, certain tests can aid in deciding what proportion of the hypertension is due to spasm and what to (apparently) irreversible pathologic changes in the vessel walls.

Unfortunate Occurrence

The development of these pulmonary changes is thus an unfortunate occurence. It is impossible to predict just which patient will develop them. The cause of pulmonary hypertension is unknown. Several unsatisfactory theories have been proposed.

1. It is a result of the high pulmonary flow itself. This fails to explain why many children and some infants develop pulmonary hypertension with ventricular septal defects, while no children but many adults with atrial septal defects develop the complication, and while only a few patients of any age with patent ductus develop it. Since patent ductus and ventricular septal defect both involve shunts from the high pressure left ventricle, pressure alone cannot be the deciding factor.

2. It represents a separate malformation. This cannot explain the gradual development of pulmonary hypertension in many patients; this development has been documented in some by serial catheterization studies.

3. It represents a persistance of the fetal thin-walled structure of pulmonary vessels, normal development to thin-walled adult vessels being prevented by the shunt. This theory, too, fails to explain the variation in frequency at different ages in different anomalies.

No Satisfactory Explanation

None of these satisfactorily explains why some individuals of identical age with identical type and size of defect will develop pulmonary hypertension and others do not.

Certain clues should arouse suspicion for pulmonary hypertension: a loud second sound in the second left interspace; certain degrees of right ventricular hypertrophy on the electrocardiogram; and diminution in vascular markings in the peripheral lung fields on the x-ray.

Small Infant

A problem of increasing importance and complexity is that of the small infant with serious, life-threatening congenital heart disease. In the first place, clinical diagnosis in an older child or adult is considerably simpler, since one can usually assume that the vast bulk of the highly complex and rarer malformations have already caused death. Such an assumption is by no means valid in the neonate.

Even in the case of a common malformation, many of the clinical manifestations are less definite and less typical in young infants. In a small baby, physiologic investigation, such as cardiac catheterization, is technically more difficult, usually involving the risks of general anesthesia, the use of less advantageous smaller catheters for the small veins, and the difficulties of hydraulic measurements, such as oxygen consumption measurements.

At the moment, the situation is not pressing, because many of the complex malformations have no satisfactory surgical repair. With the startling progress of cardiac surgery in the past ten years in mind, we can predict that this problem will become much more urgent in the future. At the present time, the chief difficulty is selecting those small infants with desperate situations who indeed have the more easily operated defects.

Summary

In summary, the decision to delay investigation and operation in a patient with congenital heart disease is fraught with deceptive pitfalls, which can be avoided only by careful and repeated evaluation based upon thoughtful consideration of the likely clinical diagnosis and the surgical techniques available.

THE PRESIDENT'S COLUMN

President Terms Southwestern Medical Meeting Outstanding

By Alvin R. Clauser, M. D., Albuquerque

I cannot refrain from again congratulating the doctors of Tucson for the wonderful convention they arranged for the 40th meeting of the Southwestern Medical Association. Those of you who were present will agree, I am sure, that the scientific program was outstanding. I can only say that those members not present missed a great convention.

Dr. Clauser

The guest speakers were well received, as evidenced by the good attendance at the lectures. An innovation this year seemed to meet with general approval. Luncheon roundtable discussions were held for the various specialties, where an opportunity was afforded to discuss the respective papers delivered during the morning sessions.

Following the luncheon the attending members were free to follow their own desires, viz: viewing medical motion pictures, golfing, swimming, sightseeing or just plain loafing and enjoying Tucson's beautiful weather.

Chuckwagon Dinner

The cocktail hour and chuckwagon dinner Thursday evening at the Saddle and Surrey Guest Ranch was delightful, the evening perfect for such an event.

Preceding the dinner dance Friday evening the Southwestern Surgical Supply Co. was host to the convention at a cocktail party. The highlight, or rather highlights, at the dinner dance was the Boys' Choir of Tucson and the jazz orchestra composed entirely of practicing physicians of Tucson. Much time and effort must have been spent to achieve such a fine orchestra. Congratulations to Dr. H. D. Cogswell and his capable muscians.

Our convention could hardly have been so successful without the presence of our ladies. It is important that they enjoy themselves at our convention. I have been informed that the ladies' activities were well planned and enjoyed by all.

I am sure that I express the sentiments of all who attended the 40th Annual Convention of the Southwestern Medical Association in saying that an outstanding program was prepared under the direction of Dr. Louis G. Jekel of Phoenix, President, and Dr. Cogswell, General Chairman, of Tucson.

Southwestern N. M., Medical Society to Meet in Deming

The next meeting of the Southwestern New Mexico Medical Society will be held in Deming, N. M., on Jan. 15, 1959, at the Deming Country Club. A panel on the subject of "Tracheotomy—General Aspects and Usefulness in Chest and Brain Injuries," will consist of Drs. W. A. Jones, E. S. Crossett, both of El Paso, and Arthur J. Fischer of Las Cruces, N. M.

W. Compere Basom, *M.D., El Paso, Orthopaedic Editor*

ORTHOPAEDIC SURGERY NOTES

Comments on Scientific Program of The 19th Annual Meeting of The American Fracture Association

Multiple pins, Smith-Petersen nail plus a firm trochanteric anchor plate can be utilized for fractures of the femoral neck and trochanteric region. The technique can be perfected to the point where multiple pins can be accurately inserted. Also a reamer can be utilized to open a hole through the neck into the head, and a cancellus plug of bone graft can be inserted across this area.

The following point was emphasized many times. No matter how strong the fixation material is, the strength of the bone determines the firmness of fracture fixation. In other words, if the bone is soft, the strong fixation can cut out and is, therefore, not stronger than weaker fixation if the bone is weak.

Accuracy Necessary

It was again apparent as it has been in so many previous occasions, that the plating of fractures must be done accurately. If screws are used across a fracture site, the proximal hole should be oversize to allow the screw to slip through and impact the opposite fragment by careful engagement. If a fracture can be treated well by plating, the plate must be well applied; and the fracture must be very accurately reduced.

The fractures which I have seen labeled as held apart by the plates actually were plated in that position. This editor believes that plates accurately applied, screws perfectly fitted; fractures carefully reduced, will give as high an incidence of union as any other type of fixation provided this fixation is used on the proper type case. Shafts of the tibia, forearm bone shafts are exceptionally well managed by this method.

Several Papers

There were several papers on fractures of the ankle, and extremely comminuted and displaced fractures were shown. Despite excellent open reduction technique some of these will have to be arthrodesed eventually. Probably some time could be saved if some of the severe ankle fractures are arthrodesed initially, if the patient understands and appreciates this opinion.

The discussion period at this meeting is always interesting and all members are urged to take part. Any member can show x-rays, photographs and so forth of problem cases and get some good advice. A well leg traction was mentioned for dislocation of the hip following the prothesis femoral head substitution operation. If the traction is maintained for about six weeks the hip may remain stable thereafter.

Fractures in Children

Fractures in children may be managed more conservatively than fractures in adults. Closed reduction, manipulation and spica type bandaging were used for supracondylars of the elbow. Adhesive traction overhead suspension for femoral shaft fractures, "T-splint" for fractures of the clavicle with complete offset revealed four years later a perfect clavicle even in a boy as old as 14 years of age. Four years later this same patient had a normal looking clavicle and originally it had a complete offset with healing in that position.

In hand injuries skin grafting over healthy surfaces, without waiting for granulation tissue to take place, usually gives better skin than that with a granulation base.

Fractures of the superior portion of the femoral head and hip joint fractures will usually give de-

generation. Probably a primary arthrodesis or femoral head prosthesis substitution is best.

Comminuted Fractures

Extremely and extensively comminuted fractures of the femur and tibia were shown treated by external pin fixation. The American Fracture Association was founded by surgeons who employed external pin fixation technique. This meeting, as has been usual in the past, had many excellent papers and exhibits showing the proper technique for pin placement for various fractures where this method is indicated. Excellent reductions and fixation were secured in extensive fractures of the shafts of the femur and tibia for instance, particularly in and through the knee joint.

It seems that if these extremely comminuted fractures are opened, the blood supply is damaged to the fragments. There is no internal fixation which works well with these, and therefore the pins externally work well. Also, it seems that if a knee joint in particular is fixed in traction so that the surfaces are pulled apart, a better range of motion is likely to result.

Beautiful Movie

A very beautiful movie revealed the technique for transplantation of the origin of the pectoralis major muscle up over the shoulder to substitute for paralysis of the deltoid.

Prolonged pain about the ankle following fractures of the os calcis or fractures about the ankle may be benefitted markedly by aspiration of the ankle where there is a little puffy swelling usually on the antero-lateral aspect and injection of hydrocortone.

In placing the Lottes nail for tibia fractures the hole in the upper tibia must be very oblique, almost in line with the intramedullary canal of the tibia. If it is not the anterior surface of the tibia may be fractured; the nail may drive out through the posterior aspect of the tibia; or the nail may bend too much on its end and come out at the lower end of the tibia anteriorly. Even compound, infected fractures have been treated successfully with this method.

Rush Pin

With the Rush pin, beautiful reduction and maintenance of position was seen in a movie with Colles' fracture in elderly people. If the styloid of the radius is intact this method is said to give good fixation.

These comments were derived from papers listed by title and author in previous orthopaedic surgery notes. Space does not allow the mention of the title and authors in this report.

All in all this meeting of the American Fracture Association, which has as its primary requisite of members that they be interested in fractures, was marked by an intense interest on the part of all the members. They participated well in the production of the program. They participated actively in the discussion periods. Fractures of the lumbar vertebrae seemed to do better by the way, if the condition is minimized and the people are treated merely with mild support, physical therapy and early ambulation and return to duty.

Pull-Out Pins

Pull-out pins for severe supracondylar fractures about the elbow in adults seemed to work well.

The 1959 meeting will be in New Orleans, the 1960 meeting will be in Mexico City. The New Orleans one will start on Nov. 1 and the one in Mexico City will start on Nov. 5.

Dr. Earle R. Pace of Santa Fe Heads Southwestern Dermatologists

New officers elected at the annual meeting of the Southwestern Dermatological Society in Tucson, Oct. 25, were Dr. Earle R. Pace, Santa Fe, President; Dr. George Waldriff, Albuquerque, Vice-President; and Dr. H. D. Garrett, El Paso, Secretary-Treasurer.

Speaker at the meeting was Dr. George C. Andrews, consulting dermatologist, Columbia Presbyterian Medical Center in New York City.

Edited By ANDREW M. BABEY, *M.D.*, LAS CRUCES, N.M.

APHORISMS and MEMORABILIA

Truths and Concepts Concerning The Nervous System

(continued)

16. "Attacks of vertigo occurring in elderly people with atheromatous arteries or suffering from chronic nephritis or arteriosclerosis must always be regarded as of serious import since they may be the precursor of cerebral hemorrhage or thrombosis."—FRENCH, *Differential Diagnosis.*

17. "We should admit the value of this sign (stiffness of the neck), but I would submit that its presence depends upon involvement of that part of the meninges which covers the dorsal aspect of the medulla. I have seen two patients with tuberculous meningitis die without any cervical rigidity —The absence of this sign, therefore, cannot exclude the diagnosis of meningitis. There are cases in which examination of the spinal fluid provides the first evidence."—C. P. SYMONDS, *Proc. Roy. Soc. Med.*, 28: 536, 1934.

Rare Case

18. "After the fourth decade it is extremely rare for any case (of migraine) to arise."—G. BRAY, *Recent Advances in Allergy*, 3rd Edit., Blakiston, Phil., p. 380.

19. "The differentiation between an upper and lower motor neuron lesion may be looked upon as the 'off tackle play' of neurology."—HAROLD R. MERWARTH, *Practical lectures delivered to The Brooklyn Hospital Interne Staff*, personal communication.

20. "It should not be forgotten that the signs of cerebellar neoplasms, particularly in children, may be precipitated by acute infection, and that an apparent complete remission of symptoms, even as long as a year, may occur in cerebellar neoplasms in children."—HAROLD R. MERWARTH (p.c.).

21. "The lancinating pains of tabes are often precipitated by acute febrile states, a finding which is peculiarly diagnostic."—HAROLD R. MERWARTH (p.c.).

22. "In syphilis of the central nervous system certain important laboratory tests are ignored by most physicians, viz., the nature and number of cells, and the quantitative content of globulin present in the cerebrospinal fluid. Accurate prognosis is dependent on these factors."—HAROLD R. MERWARTH (p.c.).

23. "The symptoms and signs of myasthenia gravis are mainly above the neck. Although uncommon, a general abnormal fatiguability may be manifest as the initial symptom."—HAROLD R. MERWARTH (p.c.).

24. "Rupture of an extracerebral intracranial arterial aneurysm is a common cause of sudden death. Many of these cases are encountered by the Medical Examiner as a result. Intracerebral hemorrhage does not result in sudden death."—HAROLD R. MERWARTH (p.c.).

25. "80% of the patients with acute Bell's palsy get well in 4-6 weeks without any treatment."—HAROLD R. MERWARTH (p.c.).

26. "It is good practice to do a gastric analysis on every case of peripheral neuritis. Polyneuritis may be the first manifestation of pernicious anemia."—HAROLD R. MERWARTH (p.c.).

Severe Headache

27. "The sudden onset of severe suboccipital headache followed by convulsions or coma in the young or middle-aged adult is often due to subarachnoid hemorrhage."— C. B. COURVILLE, *Path. Cent. Nerv. Syst.*, Pac. Press Pub. Assn., Calif., 1937, p. 325.

28. "In restless delirium following accidents with fracture of the long bones, one must always think of the possibility of fat embolism of the brain."—C. B. COURVILLE, loc. cit.

29. "The sudden onset of extreme vertigo with nausea and vomiting followed by hiccup and loss of sensibility to pain in one side of the face and the opposite side of the body speaks for thrombosis of the posterior inferior cerebellar artery."—C. B. COURVILLE, loc. cit.

ORIGINAL ARTICLES

Renal Hypoplasia*

By ROBERT F. THOMPSON, *M. D., F.A.C.S., El Paso*

Hypoplasia of the kidney is a rare malformation characterized by a failure of the organ to grow and to attain full development resulting in a miniature kidney. This tiny organ is recognizable with a distinct cortex and medulla and has a varying degree of excretory function. The condition is generally unilateral although bilateral instances have been recorded. The anomaly is estimated to occur approximately once in every 800 births.

The cause of renal hypoplasia is presumably due to developmental failure during embryological life of the 1) metanephrogenic blastema, 2) metanephric duct, 3) blood supply.

Accepted Theory

The most widely accepted theory as to the explanation of renal hypoplasia is that an inadequate and improperly distributed blood supply nourished the fetal kidney during the embryological period of development.

Herbst has suggested the possibility that there occurs faulty development of the pyramids and collecting tubules with accompanying cystic degeneration and pressure atrophy.

The unequivocal determination of the pathogenesis of a single dwarfed kidney is often impossible to achieve. The reason for this difficulty is that the end stages of renal inflammation occurring early in life may be indistinguishable from congenitally hypoplastic kidneys.

The arbitrary weight limit of 60 gm. has been set by Bell as the minimum amount of renal tissue capable of sustaining life in an adult. Within this weight limit will be included, not only congenitally hypoplastic kidneys but also instances of unilateral chronic pyelonephritis and kidneys whose blood supply has been compressed by trauma or disease.

The diagnosis may be obscured further by instances of renal infection being superimposed upon a congenitally hypoplastic kidney.

The hypoplastic kidney is usually about three to six times smaller than the organ on the opposite side which is always hypertrophic. Two types of hypoplasia have been described:

a) The architecture of the renal parenchyma is normal, with normal pelvis and ureter. This type is simply a miniature of the normally developed organ (dwarf kidney.)

b) The medullary portion and the pyramids are absent, only cortical substance being present and the pelvis is thickened and hydronephrotic.

In some of the recorded instances the microscopic studies reveal rudimentary glomeruli and tubules. In others the tissue resembles that in a normal kidney but is present in minimal amount.

In considering renal hypoplasia it must be realized that this malformation of the upper urinary tract represents an embryological defect and lack of development during fetal life when the organ, in its evolution, did not develop to its full extent or the wolffian duct of the mesonephros failed to produce a satisfactory renal bud.

Surgical Aspects

In studying the surgical aspects of congenital anomalies of the kidney it must be realized that hypoplasia is only a degree of the defective embryological process. The extreme degree of this incomplete fetal development results in a congenital absence of the kidney. The next recognizable degree of this process is renal aplasia. Renal hypoplasia is the mildest form of this congenital malformation.

The following classification and differentiation of the three related conditions are from Gutierrez:

Hypoplastic Kidney

1) Small or infantile in type; other kidney hypertrophic

*Presented at South Central Section, American Urological Association, Oklahoma City.

2) Normal renal parenchyma
   a) With medullary and cortical substance
   b) With absence of pyramidal substance
3) Microscopic sections reveal normal or rudimentary glomeruli and tubules
4) Rudimentary or hydronephrotic pelvis
5) Calyces bizarre in position and size, sometimes absent
6) Patent ureter
7) Normal urine secretion
8) Diminished or normal renal function

Renal Aplasia

1) No true kidney
2) No evidence of pelvis
3) Absence of true renal pedicle
4) Renal artery small or absent
5) Ureter incompletely developed and not patent
6) No excretion of urine
7) No renal function
8) Bladder with two normal ureteral orifices or one ectopic ureter
9) Histologic section of the supposed renal mass reveals glomeruli and tubules, showing arrest in development of renal organ.

Congenital Absence of One Kidney

No evidence of hypoplastic or aplastic renal tissue.

In renal hypoplasia the ureter is usually patent and the excretory function of the infantile organ is present to some extent. Yet this small kidney is incapable of sustaining life by undergoing compensatory hypertrophy should disease or injury incapacitate its mate on the opposite side.

It is to be remembered that the hypoplastic kidney, like all congenital renal malformations, is more likely to develop secondary diseases, particularly calculi and infection, than is the normal organ.

The principal symptom of renal hypoplasia is persistent pain in the lumbar region. However, in some cases hypertension may be the only abnormal finding which in turn produces its own clinical syndrome.

Diagnostic Procedures

The diagnosis is made by functional studies and by roentgenography. The hypoplastic kidney will invariably show diminished function both by divided renal function tests and upon intravenous urography. But retrograde pyelography will reveal the true nature of the deformity by showing a very small pelvis with absent calyces or, if it is type b, there will be seen a large, hydronephrotic pelvis with tiny renal shadow. The hypoplastic kidney usually lies lower than normal and close to the spine.

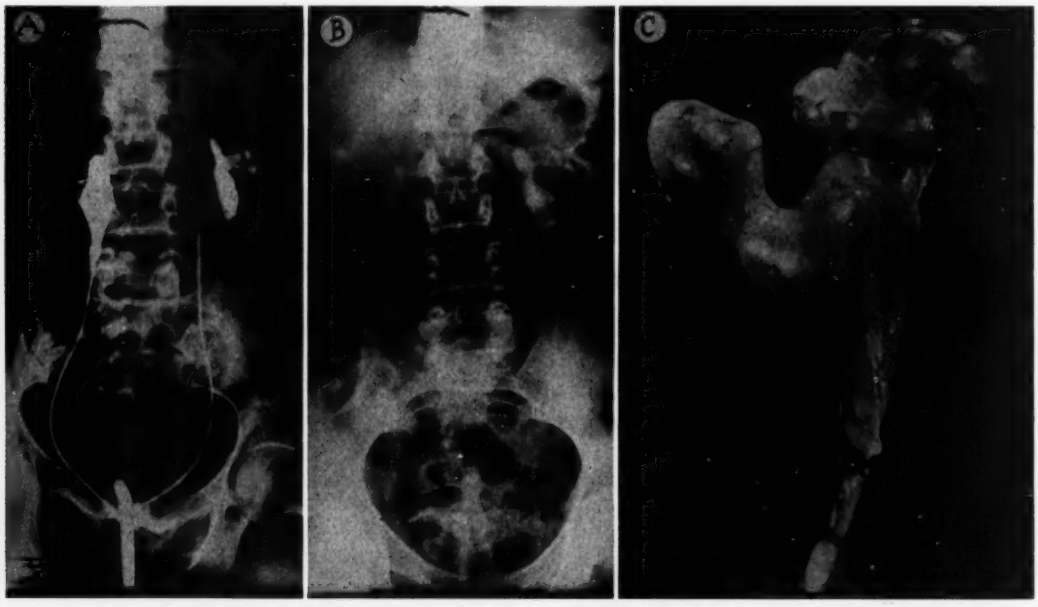

Fig. 1. A, Retrograde pyelogram shows bizarre right kidney with pelvis which is anterior and hydonephrotic; it is lower than normal and close to spine. B, intravenous urogram reveals almost complete absence of function in hypoplastic right kidney whereas function of left kidney is excellent. C, hypoplastic kidney weighs 7.6 gm. with deformed, hydronephrotic pelvis.

Renal hypoplasia should be differentiated from chronic atrophic pyelonephritis, if possible. But as stated previously, this differentiation may be extremely difficult particularly if infection has been present previously. Yet the irregular and contracted minor calyces may be in marked contrast with the smooth diminutive internal architecture of the true congenital, hypoplastic organ.

The treatment for renal hypoplasia is nephrectomy after it has been established that the other kidney has a satisfactory function.

Case Reports

Case 1. A woman aged 51 was first seen on May 3, 1956 complaining of a burning pain across her back, particularly on the right side, associated with dysuria and frequency. The symptoms had been present for about one year. She had consulted several physicians for this condition all of whom found pus and blood cells in the urine. One of them made an intravenous pyelogram which indicated that the right kidney was functionless. At this point she presented herself for complete urological investigation.

Physical examination revealed that the patient was of small stature yet healthy in general appearance. There was definite tenderness at the right costovertebral angle. A catheterized specimen of urine contained an appreciable number of pus and blood cells. Routine laboratory blood studies were normal. The nonprotein nitrogen was 35 mg. per cent. The blood pressure was 140 over 80.

Cystoscopy

Cystoscopy and retrograde pyelography revealed the pelvis of the right kidney to be anterior and hydronephrotic. There was blunting of the visualized calyces. The kidney outline was not discernible. The left kidney appeared essentially normal except that the pelvis was somewhat anterior in position (fig. 1, *A*).

Intravenous urograms revealed almost complete absence of function in the right kidney whereas there was excellent function in the left kidney (fig. 1, *B*).

Operation (May 10, 1956): Under spinal anesthesia a right lumbar incision was made and a very small kidney about the size of a plum was found with a large pelvis attached which blended into a dilated, thickened ureter. There was no definite pedicle to the miniature kidney, the blood supply consisting of several thread-like vessels attached to the organ in an unnatural manner. These vessels were ligated and divided and the pelvis was dissected free of adhesions, the miniature kidney with enlarged pelvis being removed with most of ureter.

The convalescence was uneventful and satisfactory. She left the hospital on the thirteenth postoperative day.

Following the operation she has enjoyed excellent health and has been completely relieved of the former painful symptoms.

Pathological report: "The specimen consists of a right kidney which weighs 7.6 gm. and measures 3 cm. from pole to pole (fig. 1, *C*). The capsule of the kidney strips with ease revealing a pale gray, smooth surface. The cut surface shows a renal pattern."

Microscopic examination: "The sections from the kidney show a relatively normal histological pattern with considerable hypoplasia of the glomeruli." Diagnosis: "Congenital hypoplasia of the right kidney (fig. 2)."

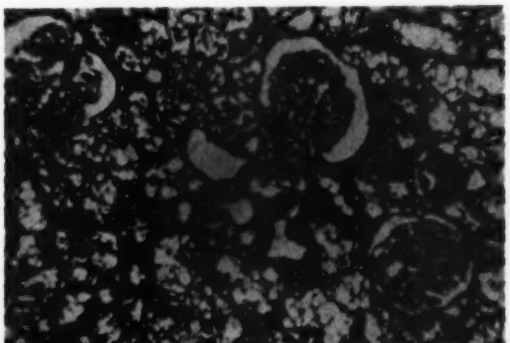

Fig. 2. Photomicrograph shows hypoplasia of glomeruli.

Case Two

Case 2. A boy aged 4 complained of a painful mass in the right lower quadrant of the abdomen. Exploratory operation by a general surgeon revealed a large, dilated, tortuous ureter. A tube was inserted in the ureter, and he was referred for urological attention.

Dye was injected through this tube in the right flank and the right kidney was seen to be small and hypoplastic with poorly developed calyces and a thin parenchyma. There was marked dilatation of the right kidney pelvis and of the right ureter (fig. 3, *A*).

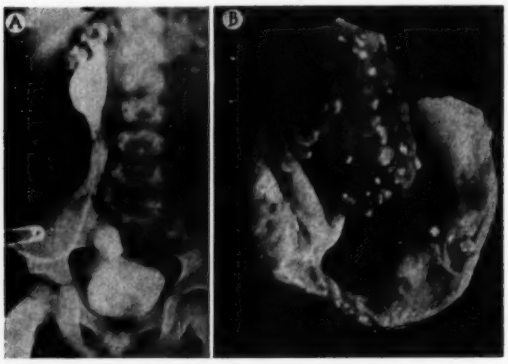

Fig. 3. A, Pyeloureterogram made by injecting tube in right ureter shows small kidney with marked dilatation of pelvis and ureter. B, small hypoplastic kidney weighs 25 gm. Note marked dilatation of pelvis and ureter.

Intravenous urography disclosed normal function of the left kidney whereas no dye was visualized on the right side, after 20 minutes. The blood chemistry was normal.

Operation

Operation (May 18, 1957): Under ether anesthesia, right nephrectomy was performed. The kidney was found to be very small to which was attached a very large pelvis and a dilated ureter. One month after the remaining ureter was completely removed through a midline incision. Six months after the last operation the child was seen to be healthy in appearance and without complaint. The urine was clear.

Pathological report: "The specimen consists of the right kidney with renal pelvis and ureter (fig. 3, *B*). The kidney measures 5 cm. in greatest diameter and weighing 25 gm. The ureter is dilated to a width of 2 cm. The serosa is covered with fibrous tags. Upon sectioning, the renal cortex is atrophic."

Microscopic examination: "The sections show that the renal cortex is atrophic. Nearly all the glomeruli are hyalinized. The interstitial tissue is heavily infiltrated with lymphocytes and plasma cells. The arteries are thickened and narrow. The ureter shows a heavy lymphocytic infiltration beneath the epithelium." Diagnosis: "Congenital hypoplasia of the right kidney with hydronephrosis and hydroureter."

Mills Building.

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CORTISONE

Cortisone In Urological Conditions With A Report Of A Trial In Interstitial Cystitis

Hoyt, H. S., J. Urol. 67:899, 1952

No definite conclusions could be reached but the author mentions an interesting point. Delayed wound healing caused by cortisone can apparently be prevented by increasing the patient's protein intake and by administration of testosterone. The latter substance offsets, to some extent, the catabolic effect of the adrenocortical hormone. (Kinsell & Margen: Unpublished data.)

Dr. A. R. Clauser of Albuquerque Named President Of Southwestern Medical Association

Dr. Clauser

Dr. A. R. Clauser of Albuquerque, was elected President of the Southwestern Medical Association at its annual meeting in Tucson, Oct. 23, through 25. The retiring President was Dr. Louis G. Jekel of Phoenix.

Other new officers are Dr. Russell L. Deter, El Paso, President-Elect; Dr. H. D. Cogswell, Tucson, Vice-President; Dr. M. D. Thomas, El Paso, Secretary-Treasurer. Members of the Executive Committee are Dr. E. W. Lander, Roswell; Dr. David Rusek, Chihuahua City; Dr. John H. Dettweiler, Albuquerque; Dr. James Fritz, Tucson; Dr. Louis G. Jekel, Phoenix, and Dr. Louis W. Breck, El Paso.

Roswell was selected as site for the 1959 meeting. The date is to be announced.

Speakers Listed

Speakers at the Tucson meeting were:

Dr. John L. Brewer, Professor of Gynecology and Obstetrics at Northwestern University; Dr. George C. Andrews, Consulting Dermatologist to Columbia-Presbyterian Medical Center in New York; Dr. John W. Henderson, Associate Professor of Ophthalmology at the Mayo Foundation in Rochester, Minnesota; Dr. Robert M. Zollinger, Professor and Chairman of the Department of Surgery at Ohio State University; Dr. Reginald H. Smart, Clinical Professor of Medicine at the University of Southern California; Dr. John R. Schenken, Professor of Pathology at the University of Nebraska; and Dr. Robert H. Lennox, Associate Professor of Pediatrics at Tulane University.

General chairmen for the meeting were Dr. Leo Kent and Dr. H. D. Cogswell, both of Tucson. Mrs. James N. Lane was general chairman of the Women's Convention Committee.

Social Events

Social events included a chuckwagon dinner at the Saddle and Surrey Guest Ranch, a cocktail hour in the Bamboo Room of the Pioneer Hotel hosted by Southwestern Surgical Supply Co., and the President's Dinner in the ballroom of the Pioneer Hotel. Providing entertainment at the President's Dinner were the nationally-famous Tucson Boys' Choir and an orchestra composed entirely of Tucson physicians as follows: Dr. Sherwood Burr, Dr. H. D. Cogswell, Dr. Harold Rosanke, Dr. Lewis Young, Dr. Wright Cortner, Dr. Wesley Soland, and Dr. S. V. Hilts.

Born in Bridgewater, S. D., Dr. Clauser received his B. S. from the University of South Dakota and his M. D. from Loyola University. He interned at Ancker Hospital in St. Paul and then practiced medicine in Canistota, S. D., for four years. In 1932 he was appointed the physician at a Madrid, N. M., coal mine. He then took a course in public health at the University of Michigan Medical School, receiving his M. S.

in public health, and in 1940 was appointed District Health Officer in Albuquerque with the State Public Health Department.

Emerges as Major

In 1942 Dr. Clauser entered the Medical Corps, served for four years and emerged with the rank of major. He next took postgraduate work in obstetrics at Tufts University School of Medicine before entering general practice in Albuquerque in late 1946.

Dr. Clauser is a past president of the New Mexico Public Health Association and a past president of the Southwestern Presbyterian Hospital staff in Albuquerque. He is a member of the Rotary Club, the First Congregational Church and the Masons. His hobbies are golf, three-dimensional photography and stereophonic music.

Dr. and Mrs. Clauser reside at 921 Avenida Del Sol NE in Albuquerque. His office is located in the Medical Arts Square. The Clausers have a son, Gerald, who is taking a pre-medicine course at the University of South Dakota.

Texas County Medical Officers Meet in Austin

One of the three major meetings held annually by the Texas Medical Association is scheduled for Jan. 24, in Austin.

It is the Conference of County Medical Society Officials and the Symposium on Legislation.

Specifically designed to help county medical society presidents, presidents-elect, secretaries, executive secretaries, delegates, and legislative chairmen to increase effectiveness of their work, the meeting is open to any other doctors who may wish to have the opportunity of hearing the outstanding speakers and attending the practical work sessions.

This coming year's program includes such formidable guest speakers as: Ernest B. Howard, M.D., Chicago, Assistant Executive Vice-President of the American Medical Association; The Honorable Allan Shivers, ex-governor of Texas; Donald E. Stubbs, M.D., Washington, D.C., Chairman of the Blue Shield Commission; and Congressman Frank Ikard, Wichita Falls, Representative from the Thirteenth Congressional District.

Other prominent persons on the program will be John McDonald, M.D., area representative for the A.M.A. Committee on Legislation; Philip R. Overton, Austin, General Counsel of the Texas Medical Association; Milford O. Rouse, M.D., Dallas, Chairman of the Texas Delegates of the A.M.A.; and James D. Murphy, M.D., Fort Worth, vice speaker of the TMA House of Delegates.

Many of the TMA's committees and councils will be meeting in conjunction with the conference on Saturday. On Sunday, a meeting of the TMA Executive Council will be held.

Last year's meeting attracted 480 persons. The day-long activity will be held at the Texas Medical Association's Headquarters Building, 1801 N. Lamar, Austin.

Scintiscanning Course Scheduled at Oak Ridge

The Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tenn., has announced that it will conduct a three-day course in scintiscanning Jan. 14-16, 1959.

The course will be an advanced review of the basic techniques as applied to the problems of radiation scanning; it will include lectures, demonstrations, and laboratory sessions, with stress placed on clinical applications, but without utilizing live clinical material. A maximum of twenty participants will be accepted from among medical doctors, their technicians, and paramedical personnel who are already engaged in doing scintiscans.

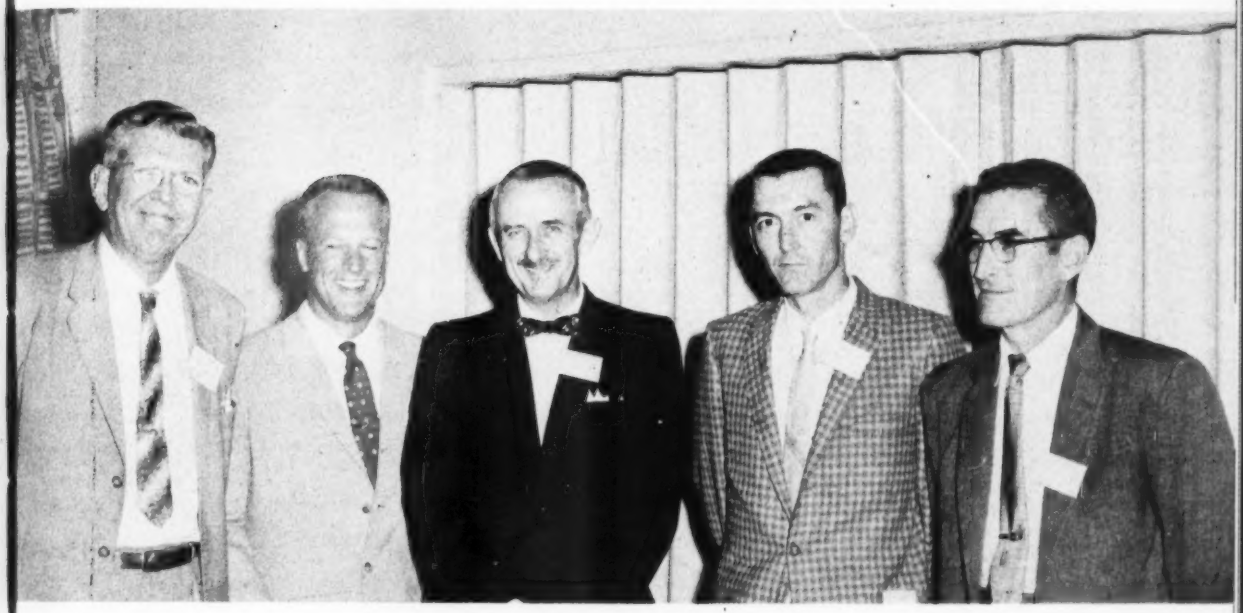
Thirty-Bed Hospital

The scanning course will be presented by the ORINS Medical Division, which maintains a thirty-bed medical research hospital, with associated research facilities, in Oak Ridge, under contract with the US Atomic Energy Commission. The Medical Division's primary concerns are with the uses of radioisotopes and radiation in medical diagnosis and therapy, mainly in the field of cancer and allied diseases.

The cost of this course will be borne under the ORINS contract with the Atomic Energy Commission; however, a nominal fee of $25 will be charged each participant, payable on the first day of the course. Applications for participation will be accepted through Jan. 2.

Additional information and application blanks are available on request from William D. Jones, Medical Division, Oak Ridge Institute of Nuclear Studies, P. O. Box 117, Oak Ridge, Tenn.

Photographic Report of the Southwestern Medical Association Meeting in Tucson, Oct. 23-25



Among new officers of the Southwestern Medical Association are, left to right, Dr. Louis G. Jekel, Phoenix, retiring president of the association and new member of the executive committee, Dr. John Dettweiler, Albuquerque, executive committee and past president of the Association, Dr. Louis W. Breck, El Paso, managing editor of SOUTHWESTERN MEDICINE and member of the executive committee, Dr. M. D. Thomas, El Paso, secretary-treasurer, and Dr. Russell L. Deter, El Paso, president-elect.

Below, shown at one of the luncheon round table meetings, are Dr. James W. Fritz, Tucson, Dr. D. Griess, Tucson, Dr. Robert Zollinger, speaker, Dr. H. D. Cogswell, Tucson, co-chairman for the meeting and new Association vice-president, and Dr. Russell L. Deter, El Paso, president-elect.

Left to right at a round table luncheon are Dr. John R. Schenken, a speaker, Dr. John I. Brewer, also one of the speakers, and Dr. George Fraser, Tucson.

Dr. Lester C. Feener of El Paso, editor of SOUTHWESTERN MEDICINE, and Mrs. Morton H. Leonard, El Paso, speculate about Lawrence Welk and the President's bo quet.

Dr. V. C. Laughlin, a visitor from Cleveland, right, chats with Dr. John W. Magee of Tucson.

Dr. Hugh Thompson, Tucson pediatrician, left, and Dr Harry Thompson, Tucson internist, get together. The Dr Thompson are no relation to each other.

Mrs. Ira Budwig of El Paso, left, and Mrs. W. A. Soland, Tucson, take a look at the program for doctor's wives.

Dr. John W. Henderson, a speaker, left, and Dr. Sherwood Burr, Tucson, take part in one of the round table luncheons. Dr. Burr is president of the Arizona Opthalmological Society.

Dr. C. E. Molholm, Albuquerque, left, and Chris Christopherson, Los Angeles, E. R. Squibb and Sons, discuss a product.

Dr. M. Chappell, Tucson, right, and Carl Anderson, Phoenix, Parke, Davis & Co.

Left to right, Dr. James Calkins, Tucson, Ray Hayden, Phoenix, and Jim Groover, Tucson, both of Burroughs Wellcome & Co.

Ade Abbott, Tucson, manager of the booming downtown development program in Tucson and a former buyer in the pharmaceutical business, visits with L. A. Roy, San Diego, Upjohn Co.

Harold D. Wilson, Tucson, left, and A. K. Hansen, Tucson, G. D. Searle & Co.

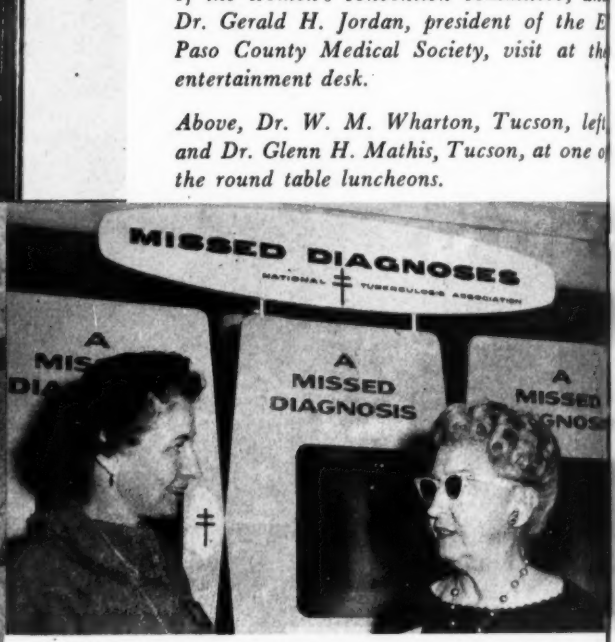
Dr. Hervey W. Dietrich, El Paso, left, and Mr. and Mrs. Harold "Wally" Walsdorf of Mission Pharmacal Co.

Left, Mrs. Hermann Rhu of Tucson, membe of the women's convention committee, an Dr. Gerald H. Jordan, president of the E Paso County Medical Society, visit at th entertainment desk.

Above, Dr. W. M. Wharton, Tucson, left and Dr. Glenn H. Mathis, Tucson, at one o the round table luncheons.

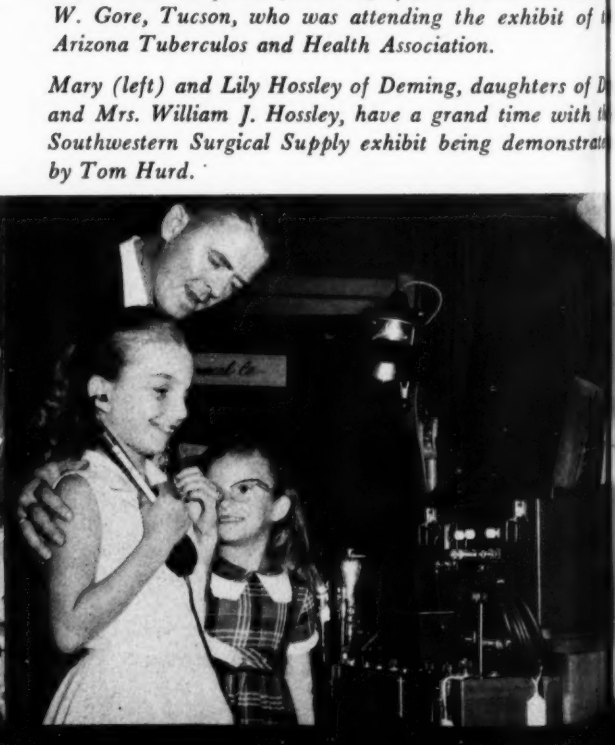
Dr. Dante Ollervides, Tampico, Mexico, left, and Dr. Victor Godoy, Peru. Both are surgical residents at St. Mary's Hospital in Tucson.

Dr. Leslie M. Smith, El Paso, a past president of the Association, left, and Dr. R. N. Class, Tucson.



Mrs. Gerald H. Jordan, El Paso, left, visits with Mrs. Ha W. Gore, Tucson, who was attending the exhibit of t Arizona Tuberculos and Health Association.

Mary (left) and Lily Hossley of Deming, daughters of D and Mrs. William J. Hossley, have a grand time with t Southwestern Surgical Supply exhibit being demonstrat by Tom Hurd.



Editors, Bless 'Em

The Presidential Address at the Annual Meeting of the Southwestern Medical Association October 23, 1958, in Tucson

By Louis G. Jekel, M. D., Phoenix

In the world of medical literature there is a battle being waged. It has been going on for years. It is the struggle between you the editor, on one hand, and you the author on the other. I say you the editor and you the author because I know that in this audience today there are a goodly number of persons who serve as medical editors in one way or another, and a larger number of persons who are medical writers at one time or another. Furthermore some of you act frequently and infrequently in both capacities. You are not strangers to the matter at hand.

In this struggle between the editors and the authors I am going to be foolish enough to try to act as a mediator. I have known the anguish of having articles of mine rejected or sent back for revision. Also I have gnashed my teeth over papers which in my opinion as an editor were totally unacceptable for publication. I think I understand some of the problems involved.

I know that no author likes to have his article rejected or cut to pieces so that he himself can hardly recognize it. I know also that no editor likes to reject or radically revise articles. In my wanderings today in this No Man's Land between editor and author I hope to point out faults on both sides. By this I do not mean to call down "a curse on both your houses."

Rather I hope to be able to tell why the editor has to do the things he does, and why the author feels the way he does. I hope to be able to help the editor place himself in the position of the author, and to help the author place himself in the position of the editor. I hope to be able to point out some things that each might do to improve matters, and in this manner I hope to help in some small way to resolve this conflict which has been going on for so long and which seems to be never-ending.

Editors are not perfect. In this statement I include not only the doctors who act as part-time medical editors, but also the professional journalists. Members of the latter group are frequently invited to criticize medical writings and medical editing. They are usually merciless, though just.

Certainly they are right in most cases, but lest they feel that they themselves are above reproach, let them read this statement which is taken from the columns of a daily newspaper in Arizona.

"Vincent Bordes, who his physician unsuccessfully sought last week to admit to a Phoenix hospital but found all crowded, now is a patient at St. Monica's, the physician said yesterday." (Arizona Republic, Feb. 19, 1948.)

These professional journalists chide us frequently on our use or misuse of words. Yet there is no unanimity of opinion in many of these cases. For instance, I recall hearing in a lecture on this subject that one of the worst habits a writer can develop is the use of negative constructions such as "not infrequently."

Yet, not an hour later I found this statement written: " . . . acquirements of the art of using words may be fundamental to one's growth and development—perhaps almost as fundamental as, and not unrelated to, the establishment of handedness." And by whom was this written? It was written by Richard M. Hewitt, Senior Consultant, Section of Publications, Mayo Clinic; Assistant Professor of Medical Literature, Mayo Foundation. There is much personal opinion in these matters.

Personally, I find these negative constructions very useful at times, and not undesirable. They are useful in expressing shades of meaning. "Not dark", for example, does not mean "light"; rather it means something between dark and light. Of course there are other means of expressing such thoughts, but I insist that at times negative constructions are not inferior to others.

Some, perhaps much medical editing is done by non-medical people. These people are usually well informed and practically always excellent

journalists. Nevertheless their lack of medical training sometimes causes them to make mistakes. Well do I remember writing once on the treatment of plantar warts with superficial x-ray therapy. I stated the dosage and listed the exact factors used, which one must always do when one is describing x-ray treatments.

The editor, however, apparently thought I was being too verbose in listing the KVP, the m.a., the TSD, and the HVL. The editor, who I am sure knows little or nothing about x-ray therapy, deleted all my factors and left me in a somewhat difficult position inasmuch as the use of my set of factors was essential if one were to use my dosage.

Admitting then that the editor has shortcomings, let me hasten to come to his defense by telling you that his shortcomings are much fewer and less important than his virtues. He can and does help the author. In his journal he will inform writers as to the style of writing used in the journal, the length of papers usually acceptable, the preparation of diagrams, the use of photographs, and the use of references and footnotes.

He will furnish proofs and help the author with his proof-reading. He will inform the author about his policies with regard to reprints. All of these things he will do in advance so that there can be no misunderstanding.

Whenever possible the editor will permit the author to employ his own style. He will not "change bad English and good medicine into good English and bad medicine." He will be honest and frank in his editing and will try to maintain friendly relations with his authors. I repeat, the editor can be and often is of great assistance to the author.

If the editors have shortcomings the authors have more. Too often papers are submitted that are not suitable for publication. The material may be unworthy, or as often happens may be submitted to the wrong journal. The writing is bad. Organization is poor or lacking, obviously due to lack of proper outlining. Paragraphs and sentences are constructed badly. Sometimes there is bad punctuation and incorrect spelling.

There are times when these papers show very poor preparation and lack of real effort on the part of the author. In some such cases one cannot blame the editor for throwing up his hands in horror and promptly issuing a rejection slip. After all, one should not expect the editor to write the paper for him regardless of how good an idea he may have had.

Just as there are certain things that the editor can do to improve matters, so are there a number of things that the author can do to help attain the same goal.

One broad statement can cover the entire matter of what the author can do to help: he can try to write well.

When a person sets out to write an article he may well first ask himself certain questions. Why is he writing this paper? He should have something to say, a message to deliver. If he has nothing to say, let him refrain from writing. Will his writing be understood? Or misunderstood?

The subject certainly should be addressed to readers who can understand it, and of course, it must be written in an understandable manner. Will his writing be read? I am sure all authors hope their material will be read.

But if there is no chance for it to be read perhaps it should not be written, although there is some justification in writing just for the sake of expressing ideas and thoughts irrespective of readers.

Having then asked himself these questions, and having come to the conclusion that he has a message, that someone will read it, that most readers will understand it and only a few will misunderstand it, the author may thereby have fulfilled the first requisite of good writing: justification in writing what he writes.

Having justified himself the author then should follow certain procedures. As a medical writer he must recognize his status as an amateur and be humble in his approach to the matter. He must learn the rules of good writing and respect them.

He certainly should put forth his very best efforts. He must strive to achieve simplicity and brevity. And finally, in writing as in all other matters, he must be strictly truthful and honest.

In his efforts to produce the best possible paper the author must re-read, revise, and re-read again. Most authorities agree that the act of revising

should extend through a period of time: days, weeks, or even months.

Revision is necessary to correct ordinary errors in punctuation, to justify the use of conjunctions, interjections, and relative pronouns, and to avoid the inconsistent use of capitals, italics, and subheadings.

In addition revision must be made to assure proper organization of material and clearness of paragraphs and sentences, to avoid repetition, and to strive for smoothness.

Unnecessary words must be removed. The summary must be a real summary, a true recapitulation of the important points in the paper. Finally, and perhaps most important of all, review and revision should be made to assure complete truth and accuracy.

Somewhere the editor and author must have a meeting of minds. Although their views may be divergent they need not be disparate. Compromise is always possible.

Each principal must make an effort to place himself in the position of the other and try to see the problem from all angles. Each must do his own job as well as he can. And each must respect the rights, duties, obligations, and responsibilities of the other. Then both may profit.

Each has great responsibilities—the editor greater, probably, than the writer. It is the duty of the editor to determine the suitability of an article for his journal. How an editor reacts to the material presented to him depends to a large extent on his own personal feelings and opinions, ideas, background, training, likes and dislikes. Also each journal has its own style and format.

Although this is largely the doing of the editor, it must nevertheless, for the sake of uniformity, be adhered to in that journal. Different journals, of course, have different styles. Therefore an article may be suitable for one journal and not for another. The editor is well aware of this. The writer should bear it in mind.

Because each journal must adhere more or less to a certain style, the editor may find it impossible to accept a paper in its original form. In such a case he may suggest changes. Most frequently these suggestions would have to do with the length of the article. Most papers are too long. If the author is willing to make such changes the editor may accept the paper as amended.

On the other hand, the author may be unwilling to make any changes. Of course he does not have to make changes, but neither does the editor have to publish the paper. Sometimes a compromise can be worked out whereby the author will agree to certain changes and the editor will agree to permit certain other things to remain unchanged.

If they fail to find an area of agreement the next move is for the author to submit his paper to some other journal and editor. This, of course, is often done.

The struggle goes on and undoubtedly will continue to go on. It would be well if it were not a struggle between the author and the editor, but rather one in which both the author and the editor would be on the same side striving to advance the science of medicine by improving the quality of the literary output of the profession.

I believe, and this I am sorry to say, that the medical author does not strive as hard for the upgrading of the product as does the editor. It is the one great goal of the latter. He wants his journal to be top-ranking and he is not to be blamed for that attitude. He wants only the best material for his journal.

He will do everything he can to attain that end, and in the final analysis he has the last word. He is the boss, and the writer must bear that fact in mind.

But remember, editors are not against authors. Editors are for themselves and their journals. They believe that what is good for them is usually good also for the writers. I agree.

So really both writers and editors are striving for what they think is right, and they both are, or at least should be, reaching for the same goal. They must co-operate. They usually do.

I like authors. I hope every one of them may find his ideal editor.

Furthermore, I like editors, bless 'em. May they rest in peace.

A Summary Report on

CORTROPHIN®-ZINC

(Corticotropin-Alpha Zinc Hydroxide)

Description: A unique patented electrolytic process (developed by Organon research) produces a complex of *alpha* zinc hydroxide and corticotropin. This complex offers considerable advantages for practical ACTH therapy.

Characteristics: New Cortrophin-Zinc provides corticotropin of unsurpassed purity with low foreign protein content. This reduces the risk of sensitization reactions.

Since about 5% of the corticotropin is uncombined, onset of clinical response is rapid. But the balance, present as a complex of alpha zinc hydroxide, provides a prolonged action so that the effective time span of a single dose is usually several days. Injection of the new electrolytic Cortrophin-Zinc is virtually painless.

Pharmacology: A potent stimulator of cortical activity, Cortrophin-Zinc *does not depress* functioning of the suprarenal glands. Unlike the corticosteroids, adrenocorticotropic hormone arouses the adrenal glands to produce *natural* steroids in natural proportions. In a 5-year study of patients on ACTH therapy, no case of adrenal *or pituitary* depression or atrophy has been observed.

Because Cortrophin-Zinc is virtually painless on injection and its prolonged action obviates frequent injections, it is now practicable to use Cortrophin-Zinc in most of the indications where formerly reliance has been on corticosteroids. This freedom from apprehension of deleterious depressive effects permits clinical use of valuable hormone therapy on a broader scale than has been possible heretofore.

Clinical Uses and Dosage: The many published reports on the use of Cortrophin-Zinc as well as ACTH, in thousands of patients indicate its value in over 100 disorders. Most responsive have been: allergies and hypersensitivities, rheumatoid arthritis, bronchial asthma, serum sickness and inflammatory skin and eye diseases.

Dosage should be individualized, but generally initial control of symptoms is obtained with a single injection of 40 units of Cortrophin-Zinc daily, until control is evident. Maintenance dosage is generally 20 units (or less) twice a week.

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MONTHLY CLINICAL PATHOLOGICAL CONFERENCE
EL PASO GENERAL HOSPITAL

September 18, 1958

FREDERICK P. BORNSTEIN, M. D.—*Editor—Case No. 1060*
Presentation of case by Dr. Jack Postlewaite

History:—Dr. Nathan Kleban:

A 53-year-old negro housewife was admitted to the hospital on May 15, 1958. For two weeks the patient was unable to eat, bled from her rectum and vomited blood before admission. Another history recorded the rectal passage of dark blood and liquid stools without abdominal pain. The medical consultant's note stated that there had been dark, loose, foul-smelling stools of tarry consistency.

The patient had two children. Menstrual flow had stopped about 1948. Total hysterectomy, bilateral salpingo-oophorectomy and appendectomy was performed in another hospital in March, 1953. A doctor had treated her for hypertension.

Father and mother were dead of unrecorded cause.

Physical Examination:

T. 99 P. 98 R. 24 B. P. 138/80.

The patient showed slight confusion. She was obese. The cardiac PMI was in the left sixth intercostal space. There was epigastric tenderness with radiation to the right lower abdominal quadrant. No masses were felt on digital rectal examination.

Hospital Course:

When she reached the ward the patient complained of abdominal pain. Hematocrit was 14 volume per cent, hemoglobin 4.8 grams per cent; WBC 11,600; segs. 85 and lymphs. 15. Hematocrit rose to 23 after two units of whole blood. Urine was straw in color, acid, cloudy, 1.013, albumin 1+, sugar negative, loaded with pus cells and WBC clumps.

The patient was given a Sippy diet, reserpine, vitamin K, Andrenosem, antacid, and Gelfoam powder dissolved in milk.

Duodenal ulcer niche and gastro-duodenitis were reported following an upper GI series on the second day. Two additional units of blood raised the hematocrit to 30. Temperature rose to 100 on the second and third days but did not exceed 99 again until the day before death. Recorded blood pressures ranged from 142 to 220 systolic and 90 to 120 diastolic. On the third day the fifth and sixth units of whole blood raised the hematocrit to 39. Nurses' notes said that the patient was restless, moaned and groaned, complained of weakness and hunger. Small emesis occurred once.

Bland Diet

Diet was changed to bland on the fourth day. Kaopectate with Neomycin was prescribed because of 11 loose stools. On the following day the patient felt better, had only one small dark brown liquid stool. Hematocrit was 41 the next morning, the sixth day, when a soap suds enema returned small particles of green fecal matter having a foul odor.Tap water enemas were then given until returns were clear in preparation for sigmoidoscopy. A cause for rectal bleeding was not seen during the examination. Additional tap water enemas were given that night in preparation for a barium enema. This was followed by rectal bleeding.

No defects were seen with barium enema up to the recto-sigmoid. Satisfactory visualization beyond that point was not obtained because the patient was unable to retain the barium. APC tablets were prescribed for pain.

The next morning, the eighth day, the patient refused breakfast, was restless and uncooperative, later was listless and somnolent. Hematocrit was 38; WBC 16,300: seg. 81, stabs. 1, lymphs. 8, eosinophils 1 (only 91 cells reported). Scheduled surgery was cancelled.

Patient Restless

On the ninth day the patient was again restless, uncooperative, insisted on going to the bathroom, began scratching her buttocks and rectum. Sparine was administered and her hands were restrained. A small amount of rectal bleeding was noted. Oral intake was poor and continued so. Sitz bath was given the next day for apparent infection around the rectum. Urine was yellow, clear, acid, 1.013, albumin 2+, sugar negative, WBC too numerous to count, rare hyaline cast. Sparine was again given for restlessness. Later the patient

was stuporous but shouted and yelled at intervals. Uremic frost was noted by the nursing staff.

Normal saline soaks were applied to the rectum, from which there was foul drainage. Coma succeeded stupor. Rectal temperature rose to 105. A portable chest X-ray picture was interpreted as demonstrating clear lung fields, cardiac enlargement with prominence of left ventricular segment, and elongation, tortuosity and dilatation of the aorta. Conclusion was findings consistent with arteriosclerotic and hypertensive cardiovascular disease. An accompanying aneurysm of the aorta cannot be excluded. Serological test for syphilis was negative. 2500 cc. of 5 per cent glucose/D.W. was given I. V. Blood pressure dropped to 138/70. A foul smelling fluid drained from her mouth.

Nitrogen Rises

Blood urea nitrogen rose from 50 to 71. Nonprotein nitrogen was 130. Other chemistries reported calcium 7.3 mg. per cent, CO_2 capacity 22.7 mEq/L; chlorides 160 mEq/L; potassium 9.0 mEq/L; sodium 191 mEq/L. Hematocrit was 37.

There was involuntary stool, later rectal passage of what seemed to be old blood. Brown liquid material was vomited and suctioned. Shock persisted. Respirations were labored, then gasping and Cheyne-Stokes in pattern. Urine output was 500 cc. on the day before death, 150 cc. on the last day. No fluid was given. Nurses' notes described the patient as appearing dehydrated. A pericardial rub was heard. Death occurred 11½ days after admission.

Clinical Discussion—Dr. Jack Postlewaite:

It seems to me that we are dealing with a problem that has been solved already on paper. Evaluating this problem is merely trying to bring out the more cryptic, esoteric diagnoses that are famous at this institution. We are now in a position to make a diagnosis because we are not only unlimited in my personal imagination but the history and physical contribute nothing. So we will proceed with the esoteric diagnoses.

This should in all due respect be nothing more than a colored woman who had symptoms of a duodenal ulcer. She bled both directions, orally and rectally. She was a known hypertensive, she had renal disease which terminated in uremia. Her demise was due to uremia with potassium retention. Obviously we will have to find something more interesting than this sort of classical situation to account for our findings.

She is 53 years old. She is a colored female. Therefore, we ought to talk about sickle cell anemia. They do bleed. The patient was confused throughout her clinical history. Something was wrong with her head but we could not account for it on admission. She had 4.8 grams hemoglobin and a hematocrit of 14. The white count wasn't particularly significant, nor the differential, 85 per cent polys and 15 per cent lymphs. She was slightly febrile and dehydrated.

On admission she apparently had evidence at least suggesting rectal bleeding and the history stated oral. It was painless abdominal bleeding. One of the conditions, which lead to fatal termination and which are hard to discover clinically, is pancreatitis; but it is associated with painful, not painless, bleeding. I don't recall pancreatitis ever being painless. Except on examination did this lady have pain and remember she was confused, so her history may not be entirely reliable? This pain was epigastric relating to the right lower quadrant.

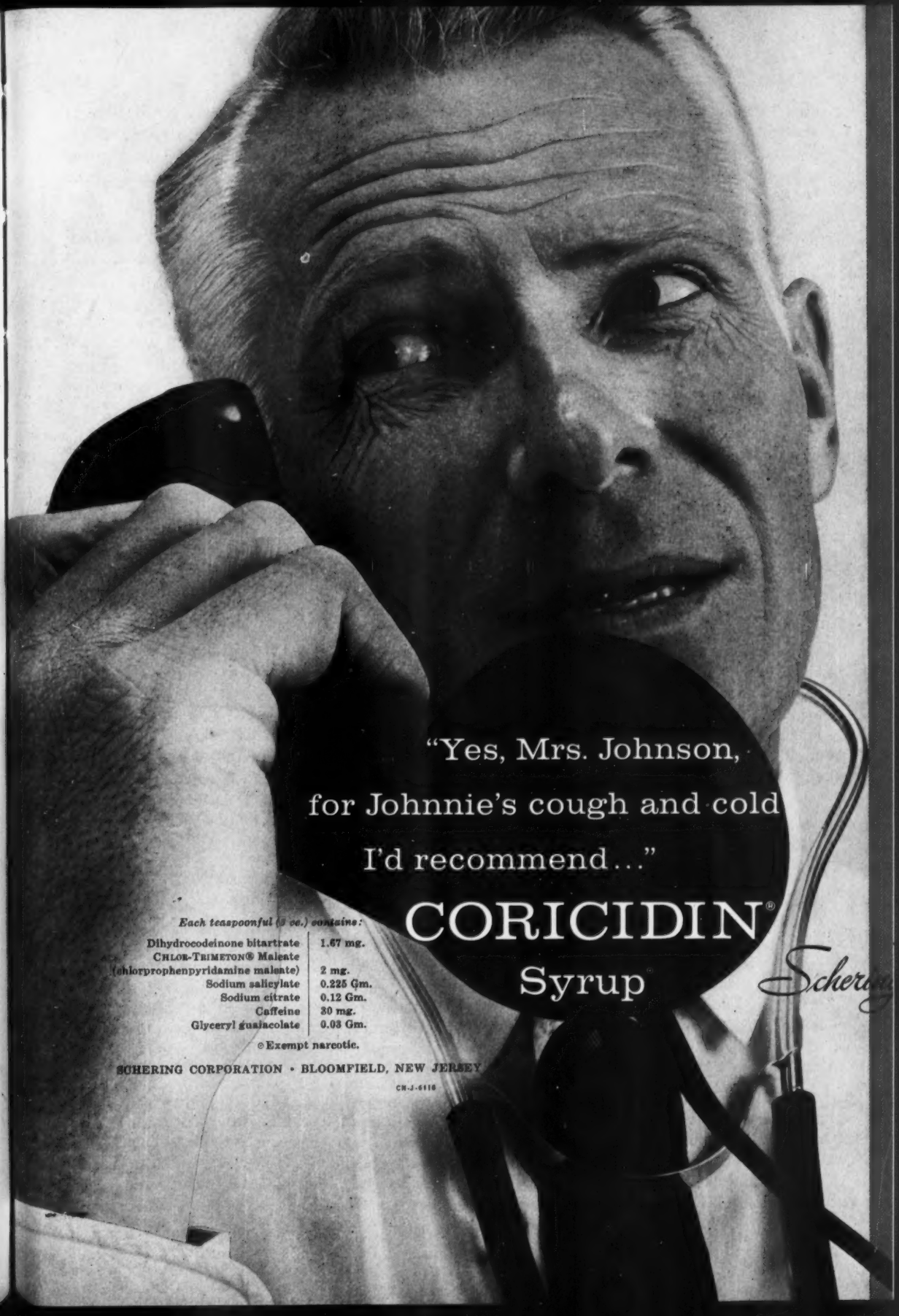
She had nothing of importance on history other than hypertension. On laboratory studies the anemia was apparent. Replacement apparently did considerable good because she doubled her hematocrit with two transfusions. The urinalysis: The specific gravity on two occasions—this lady was apparently repeatedly dehydrated—was 1.013. Albumin one and two plus and loaded with pus cells. On one occasion a few hyaline casts were found. Now this adds to our renal picture.

Progressive Uremia

At least on admission one wants to know why the amount of pus in the urine until you get on with the story and discover that she develops a progressive uremia. The blood urea nitrogen started somewhere around 50 and ended up to 71 mg. per cent. The NPN is reported 130. The other interesting phenomenon is the evaluation of the white count which goes with the gastro-intestinal bleeding.

She also developed this weird and complex electrolyte picture which may be due to replacement plus retention. Nevertheless we are told she had a potassium of 9 mEq/L. The other interesting finding is the blood calcium of seven mg. per cent.

Now to evaluate this complexity of history physical and laboratory data; The woman took 12 days approximately to die. She apparently died of a terminal uremia. She had some sort of a bowel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disorder manifesting itself in GI bleeding, oral and rectal. She also had a diarrhea for which she received antibiotics. It is interesting we don't know more about the stool, perhaps a clue would have been obtained from its study.

Major Categories

We then should approach the problem by trying to see how these facts fit in with the major categories of pathology that we see as clinicians.

First, infection. One can't account for this entire problem on the basis of infection, but certain organs certainly show a response to infection or irritation, such as the kidney and the GI tract. I suppose an ulcer is an inflammatory process as well as a degenerative disease. There is a vascular element. Ulcers have an inflamed wall and the ulcer that penetrates may perforate. It is unusual for the GI system to develop diarrhea, bleeding and and so forth and not show a good deal of pus. I don't think it is an inflammatory GI lesion. She is hypertensive. I don't know of any illness here that should be inflammatory. Her cerebral symptoms are secondary to her general body debility.

Degenerative: Does she have an aneurysm? Well, a hypertensive patient has at least an altered blood vessel wall. There is nothing inflammatory here. Her serology is negative and I don't see that the circulatory system could be incriminated on an inflammatory basis but it may well be on a degenerative basis. She is entitled to a dissecting aneurysm. There is no reason why some of the blood couldn't have been lost by dissection, and perhaps this cut off her renal system. Perhaps this also gave her alteration of the central nervous system.

Possible Shrunken Kidney

It may be this kidney is nothing more than a shrunken kidney. It would be pyelonephritis which is certainly by far the most common chronic disease of females. It could be the arteriolitis groups, nephroarteriosclerosis because she is a chronic hypertensive. Thus her uremia was tipped over when she developed her GI hemorrhage with re-absorbed blood, etc. So the degenerative diseases are important in this case.

Neoplastic: I am sorry we have no way of going after that. It does take tissue to make that diagnosis, at least some kind of a shadow that is suspicious, so that we will dispense with neoplastic diseases although the possibility exists. Metabolic processes are involved, and those are the natural consequences of aging. There is no evidence to consider diabetes. At least we see no sugar. She is obese and it makes one wonder if it wasn't some sort of apoplectic process of the pancreas, etc. I don't know why they did a calcium on her other than that she was uremic. This is a good investigation for all the things that go wrong in uremia. Now the other classifications of trauma and congenital disease are out.

Positive Findings

On analysis of the positive findings this problem fits best our degenerative types of disease. Degeneration and necrosis are like processes, or steps from one to the other. One can throw in a weird disease which I enjoy doing before this group, the collagen group. She has got the multiple systems involved.

There is nothing to suggest that her hypertension contributed to this demise. There is nothing to be sure of other than the X-ray saying a niche in the duodenum suggested an ulcer. Is this the only source of GI bleeding? There is a great deal of evidence of sigmoidoscopic examination that she doesn't have any diffuse bleeding.

She does not have an ulcerative colitis or diffuse hemorrhagic colitis. So one might consider then some fulminating degenerative process, collagen group, affecting kidneys, bowel, vascular tree, sensorium, etc. But I have no clue to this and there is no tissue which was studied including blood and bone marrow. It seems then we will resort back to the simple diagnosis, she had an ulcer, she was not uremic on admission, she bled, vomited blood, etc.

End Stage Kidney

Replacement was perfectly satisfactory but she had an end stage kidney, probably pyelonephritis. She didn't bleed again in the hospital. Transfusion was excellent. She managed to keep her blood. There was surgery planned along the way. I assume they were going in to tie off bleeders or remove ulcers or explore, and I suspect that the exploratory lap was delayed because she became critically ill again on the floor.

It occurs to me that the laparotomy might have been most revealing, that she came in with a simple ulcer, had a uremic process in progress and something of an apoplectic type happened in the hospital. Those things include myocardial infarctions. They include dissection of vessels, blown

out vessels, hepato-renal syndrome, liver shut-down. We can go on with a maze of possibilities. This is a GI bleeding due to ulcer, uremia due to end stage kidneys, pyelonephritis and the terminal event was the uremia.

Dr. Bornstein:

In-asmuch as Dr. Postlewaite suggested some surgery was planned, do any of the surgeons feel that they would like to comment on the diagnosis and management of this case?

Dr. Dotson:

The reason why we postponed the operation was her condition which deteriorated rapidly. Our impression at the time was that of a peptic ulcer which we were going to treat. It seemed to me that if she recovered enough in the first few days, we felt we could explore her. Then she developed convulsions and erratic behavior and semi-stupor on the ward. We took a BUN and found it to be 70 or so. It hadn't been taken previously to that time. She went rapidly on to her demise and so far as I know it was chronic kidney failure with superimposed massive GI hemorrhage as Dr. Postlewaite suggested.

Dr. Haskell:

I was wondering about the treatment the patient was given—Sippy diet and reserpine. Do you people ever have trouble with ulcers without a previous history when you give them reserpine? It seems to me that could be contra-indicated in a case like this and also the vitamin K. Did you have any indication that she had some liver damage?

Dr. Dotson:

I think we just assumed that she had chronic kidney disease aggravated by the shock of the hemorrhage. She was not in shock on admission. Evidence of kidney disease was not suspected at all except when I saw her the day before surgery. She was exhibiting mental confusion and I noted at the time the low specific gravity of the urine. For that reason I ordered a BUN.

Dr. Licon:

Merely as a casual glance at this protocol I note that serological test for syphilis was negative.

Dr. Hassler:

The general consensus is that she died of an end stage kidney. Well, exactly what is an end stage kidney? You mean she had a lower nephron nephrosis when she came in secondary to the shock or what?

Dr. Postlewaite:

If a man is lucky enough to live long enough with these chronic diseases, everything becomes end stage. What I had in mind, however, is the end stage as the pathologist uses the term. Having been exposed to it several years here, I use his terms more than I do mine. An end stage kidney is a small, contracted kidney. The arteriolarnephrosclerosis, the malignant arteritides don't fall in this group. The chronic nephrosclerosis group develops small and contracted kidneys.

All inflammatory kidneys will become small and contracted when they become chronic pyelonephritis or chronic glomerulonephritis. These are just small and contracted kidneys and they give us an awful time clinically because they become salt-losing kidneys and we are sitting here with salt restriction until the patient is rushed to the hospital because they fall on the floor every time they stand up. We think it is the reserpine, etc., which I am certain didn't do that, and so we take them off the hypotensive agents and we find out they don't have any salt left in their bodies.

They are putting out tremendous amounts of urine which they didn't do all the 10 years before we saw them, so these are salt losing kidneys, end stage, both functionally and clinically, and they may carry uremia a long time and not be tipped over into the mortal statistics until something else happens. I don't know that she is salt-losing. She certainly developed anuria. I suppose the urologist would like to add papillitis and dozens of other things that happen to kidneys that stop, but at the moment that is my answer.

Clinical Diagnosis: Bleeding peptic ulcer.

Dr. Postlewaite's Diagnosis: Chronic pyelonephritis with uremia.

Pathological Diagnosis: Chronic pyelonephritis with uremia, and nephrolithiasis.

Pathological Discussion: Dr. Bornstein:

On autopsy there was a rather obese woman. The heart was moderately enlarged, weighing 400 grams, suggesting that there was hypertension of some duration. The stomach and duodenum were filled with large amounts of hemorrhagic fluid. Careful examination of the mucosa of the stomach and duodenum revealed numerous petechial hem-

orrhages. However, no definite ulcer or ulcer scar was demonstrable. The pancreas was not remarkable. The liver and spleen were not remarkable.

The left kidney weighed 75 grams, which is small. The right kidney weighed 120 grams which is also a small kidney. The difference in size between the two kidneys implies a pyelonephritis type of process. In addition the smaller kidney contained a large stone. The smaller kidney was finely and irregularly granular with some deeper depressions on the surface. In addition this kidney had a so-called "flea-bitten" appearance which used to be connected with the term malignant nephrosclerosis. I will talk about that term later. The two kidneys show essentially the same abnormalities.

Atrophy of Cortex

The cut surface was mainly remarkable for the atrophy of the cortex and the fact that the renal pelvis was pale and not involved in an active inflammatory process. Microscopically there was uniform and severe destruction of the renal parenchyma. The small arterioles showed the so-called onion skin pattern of obliterative arteritis. The tubules were distended and filled with inflammatory cells.

The glomeruli were not too bad. Some of them show hyalinization but the main disease process was limited to the sclerosed arterioles and the tubules which were filled with lymphocytes and plasma cells. This, then, in the true sense of the word, is the end stage kidney, chronic Bright's disease, but I think we can go a little bit further in this case in trying to explian what has happened to this kidney.

I think the chronic glomerulonephritis here is out. The kidney is a kidney which used to be called malignant nephrosclerosis which is actually a clinical term. Weiss and Parker pointed out that chronic pyelonephritis will give you a picture of where your main damage is in the tubules and in the arterial and interstitial system.

Chronic Inflammation

Any chronic inflammation, whether it is in the kidney or gall bladder or anywhere else, will be accompanied by a long standing endarteritis which we have here. You have a diffuse, far reaching destruction of both kidneys which I think is responsible for the uremia and we see very often that intestinal hemorrhages occur with a uremia. I think essentially then this woman died with kidney failure with accompanying hemorrhages.

When Vollhard and Fahr first brought out their classification they held that the arteriolar destructive process was the primary damage in the kidney, but I think since the work of Weiss and Parker we believe much more than chronic pyelonephritis is responsible for this type of kidney destruction. They all end up in uremia and die from uremia, and I think that is what happened here.

Dr. Licon:

Was there any syphilis in this case?

Dr. Ayub:

Can I ask why, in some of these end stages, you have a very, very low red count and there has been no significant bleeding?

Dr. Bornstein:

There is no evidence of syphilis in this case. The question is what is the mechanism of the severe anemia in end stage kidneys and uremia. The answer is fairly simple—there doesn't seem to be any. About two years ago Limarzi from the University of Illinois published a paper on about 200 cases of uremia that were followed at the Illinois Research and Educational Hospital, with bone marrow biopsies. He came to the reluctant conclusion that he had no explanation for the mechanism of anemia in chronic nephritis and uremia.

Dr. Postlewaite:

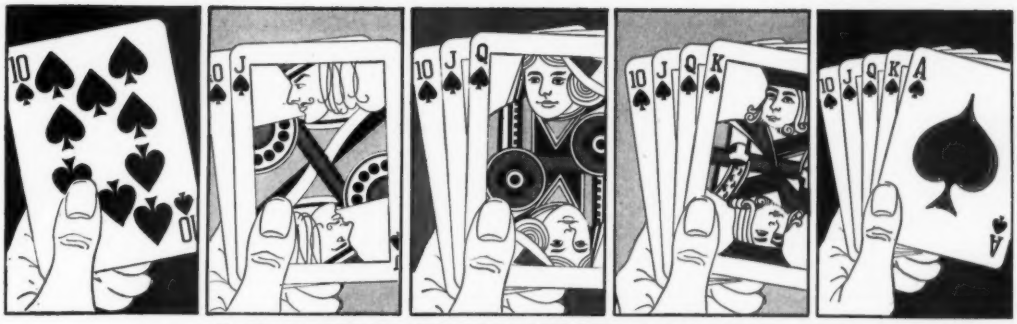
I have got news for you. You are about two years behind. The kidney is the producer of an erythropoietic hormone.

Dr. Bornstein:

The trouble with that observation is the fact that the bone marrows don't show any depressed erythropoiesis in these cases.

Dr. Kleban:

It is true that the exact cause of anemia in uremia is not certainly known. There is at times and there has been demonstrated at times a hemolytic factor and there has also been demonstrated bone marrow depression and apparently in different instances either or both of those factors may play a part. The use of reserpine in individuals with an ulcer or gastroenteritis: Certainly this may contribute to the development or to the exacerbation of an ulcer. In this particular instance the house staff was concerned when her blood pressure became elevated.



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Her hospital course would indicate that actually she had no serious bleeding after she came to the hospital. Her hematocrit was raised to 41, remained between 37 and 41. The factor that I would like to point out has to do with what I think actually was her precipitating cause of death.

Fundamental Difficulty

I don't disagree that her kidney disease was her fundamental difficulty and that it was probably made considerably worse by the hemorrhage, with the resulting decrease in renal blood flow. In December of 1956 there was a patient presented here who had gallstone ileus with a gallstone in the ileum resulting in a mechanical obstruction and peritonitis.

In this particular case this woman was given castor oil in preparation for barium enema. Four hours later she became clammy, restless, and was given seconal. Her blood pressure at that time was 80/60. She was mentally confused, she was then given tap water enemas as further preparation for barium enema and during this process she died. The patient who is being discussed tonight actually was doing fairly well on her fifth hospital day.

On the fourth day she had diarrhea, 11 loose stools. The color was not described. On the fifth day she had one brown stool. On this day she was given enemas in preparation for sigmoidoscopy and in preparation for barium enema.

Mental Confusion

Following this there developed mental confusion. Following this there was virtually no oral intake for the remaining seven days of her life. It is my feeling that the repeated enemas that she received on this particular day apparently depleted her of fluid and electrolytes to a critical point. An individual with severe renal disease on the threshold of irreversible failure, can tolerate no further diminution of renal blood flow.

One must be careful, for example, in preparing an individual with elevated nitrogen for intravenous pyelogram. A laxative plus an enema will produce sufficient depletion of extracellular fluid to drop the effective blood flow. In turn there will be a drop of renal blood flow and this just may make sufficient difference to throw the patient into irreversible renal failure.

Dr. Bornstein:

Let me say one thing. I think when it comes to the question of anemia associated with uremia we should be careful to distinguish what is an appetizing hypothesis and what is fact. If you know that there is an erythropoietic hormone in the kidney and this is responsible, you should find a consistent bone marrow depression in uremia which you don't find.

If you assume that there is a hemolytic factor responsible for the anemia in uremia you must remember that a hemolytic anemia severe enough to do damage will show you visible hemosiderin in the spleen and in the bone marrow. You don't have it. So for the time being you have to charge this up to appetizing hypothesis. The facts that permit us to say what causes uremic anemia are not in at the present time.

Coming Meetings

Southwestern New Mexico Medical Society, Deming Country Club, Deming, N. Mex., Jan. 15, 1959.

University of Colorado Medical Center, Denver, Colo., Postgraduate Course, General Practice Review, Jan. 19-24, 1959.

American College of Surgeons, Sectional Meeting, Shamrock Hilton Hotel, Houston, Texas, Feb. 2-4, 1959.

University of Texas Postgraduate School of Medicine, Temple Division, Seventh Postgraduate Conference in Medicine and Surgery, sponsored by Scott, Sherwood and Brindley Foundation, Temple, Tex., Mar. 2-4, 1959.

University of Colorado Medical Center, Denver, Colo., Postgraduate Course, Medical Technology, Mar. 16-21, 1959.

Biennial Western Conference on Anesthesiology. Approved for 24 hours Category II Credit, A.A.G.P. Westward Ho Hotel, Phoenix, Apr. 1-4, 1959. For information write to Boyden L. Crouch, M.D., 301 W. McDowell Road, Phoenix, Ariz.

The Arizona Medical Association, 68th Annual Meeting, San Marcos Hotel, Chandler, Ariz., April 29-May 2. Friday, May 1, has been designated as Medical Education Day.

New Mexico Medical Society, annual meeting, Las Cruces, N. Mex., May 5-7, 1959.